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FILE 'HOME' ENTERED AT 16:43:43 ON 07 MAY 2001

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FILE 'MEDLINE' ENTERED AT 16:43:47 ON 07 MAY 2001

FILE 'CANCERLIT' ENTERED AT 16:43:47 ON 07 MAY 2001

FILE 'BIOSIS' ENTERED AT 16:43:47 ON 07 MAY 2001

FILE 'EMBASE' ENTERED AT 16:43:47 ON 07 MAY 2001

FILE 'SCISEARCH' ENTERED AT 16:43:47 ON 07 MAY 2001

=> s protein kinase b or akt

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   3 FILES SEARCHED...
L1          7156 PROTEIN KINASE B OR AKT

=> s l1 and (cardiomyo? or cardiac)

L2           212 L1 AND (CARDIOMYO? OR CARDIAC)

=> s l2 and py<=1998

   2 FILES SEARCHED...
   3 FILES SEARCHED...
L3            25 L2 AND PY<=1998

=> dup rem l3

PROCESSING COMPLETED FOR L3
L4            10 DUP REM L3 (15 DUPLICATES REMOVED)

=> d ibib abs 1-10


L4   ANSWER 1 OF 10  MEDLINE                                  DUPLICATE 1
ACCESSION NUMBER:    1998380465      MEDLINE
DOCUMENT NUMBER:     98380465   PubMed ID: 9712868
TITLE:               Prostaglandin F2alpha (PGF2alpha) and the isoprostane, 8,
                     12-iso-isoprostane F2alpha-III, induce
                     cardiomyocyte hypertrophy. Differential activation
                     of downstream signaling pathways.
AUTHOR:              Kunapuli P; Lawson J A; Rokach J A; Meinkoth J L;
                     FitzGerald G A
CORPORATE SOURCE:    Center for Experimental Therapeutics, University of
                     Pennsylvania, Philadelphia, Pennsylvania 19104, USA.
CONTRACT NUMBER:     DK44730 (NIDDK)
                     DK45696 (NIDDK)
                     T-32-HL07843-02 (NHLBI)
                     +
SOURCE:              JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Aug 28)
                     273 (35) 22442-52.
                     Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY:        United States
                     Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199809
ENTRY DATE:          Entered STN: 19981006
                     Last Updated on STN: 20000303
                     Entered Medline: 19980924
AB   Prostaglandin receptors may be activated by their cognate ligand or by
     free radical catalyzed isoprostanes, products of arachidonic acid
     peroxidation. For example, prostaglandin F2alpha (PGF2alpha) causes
     hypertrophy of neonatal rat ventricular myocytes, via the PGF2alpha
     receptor (FP). However, the FP may also be activated by the isoprostane,
     8,12-iso-iPF2alpha-III (Kunapuli, P., Lawson, J. A., Rokach, J., and
     FitzGerald, G. A. (1997) J. Biol. Chem. 272, 27147-27154). Both ligands
     induce myocyte hypertrophy with overlapping potencies. Interestingly, the
     hypertrophic effects of these two agonists on cardiomyocytes are
     additive. Furthermore, the preference of these two agonists for
activation
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of intracellular signal transduction pathways differs in several respects.

Thus, PGF2alpha and 8,12-iso-iPF2alpha-III stimulate inositol phosphate formation with EC50 values of 50 +/- 12 nM and 3.5 +/- 0.6 &mgr;M, respectively. Moreover, PGF2alpha causes a robust activation (approximately 50-fold) of Erk2, whereas 8,12-iso-iPF2alpha-III has no effect. Similarly, PGF2alpha causes translocation of cytosolic phospholipase A2 and also results in a 7-fold increment in the formation of 6-keto-PGF1alpha, whereas 8,12-iso-iPF2alpha-III exerts no effect on this pathway. On the other hand, both agonists are equally potent in activating JNK1 and c-Jun, whereas neither activates the p38 kinase. Both PGF2alpha and 8,12-iso-iPF2alpha-III activate the p70S6 kinase (p70(S6K)),

but not **Akt**, downstream of phosphatidylinositol-3-kinase (PI3K).

However, both wortmannin, a PI3K inhibitor, and rapamycin, an inhibitor of

p70(S6K) activity, inhibit 8,12-iso-iPF2alpha-III -induced myocyte hypertrophy, with IC50 values of 60 +/- 12 and 3 +/- 1.7 nM, respectively,

whereas neither compound abrogates the PGF2alpha-mediated response. Thus, both PGF2alpha and 8,12-iso-iPF2alpha-III induce myocyte hypertrophy via discrete signaling pathways. Although both agonists signal via the JNK pathway to initiate changes in c-Jun-dependent gene transcription, PGF2alpha preferentially activates the MEK-Erk2- cytosolic phospholipase A2 pathway. In contrast, the PI3K-p70(S6K) pathway appears to be essential

for 8,12-iso-iPF2alpha-III-induced myocyte hypertrophy.

L4 ANSWER 2 OF 10 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998212002 MEDLINE
DOCUMENT NUMBER: 98212002 PubMed ID: 9545305
TITLE: Activation of phosphatidylinositol 3-kinase through glycoprotein 130 induces **protein kinase B** and p70 S6 kinase phosphorylation in **cardiac** myocytes.
AUTHOR: Oh H; Fujio Y; Kunisada K; Hirota H; Matsui H; Kishimoto T;
Yamauchi-Takihara K
CORPORATE SOURCE: Department of Medicine III, Osaka University Medical School, Suita, Osaka 565, Japan.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1998 Apr 17)** 273 (16) 9703-10.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980529
Last Updated on STN: 19980529
Entered Medline: 19980521

AB Phosphatidylinositol (PI) 3-kinase is known to be activated by cytokine stimulation through different types of receptors to transduce intracellular responses. We have previously reported that leukemia inhibitory factor (LIF) induces the activation of Janus kinase signal transducer and activator of transcription (JAK-STAT) and mitogen-activated

protein (MAP) kinase pathways through glycoprotein (gp) 130 in **cardiac** myocytes. However, whether PI 3-kinase is involved in

regulation of gp130 signaling and the activation mechanisms by which it associates with other tyrosine-phosphorylated proteins remain unknown. We found that LIF induced the activation of PI 3-kinase in **cardiac** myocytes. Moreover, JAK1 binds to PI 3-kinase, and LIF stimulation increases the PI 3-kinase activity in JAK1 immunoprecipitates. Activation of MAP kinase and **protein kinase B** by LIF was attenuated by wortmannin. LIF-induced p70 S6 kinase activation, protein synthesis, and c-fos mRNA expression were inhibited by wortmannin and rapamycin. Both inhibitors failed to appreciably affect the phosphorylation of STAT3. In conclusion, PI 3-kinase is activated with LIF in **cardiac** myocytes, and JAK1 is found to associate with this enzyme. PI 3-kinase provides a crucial link between gp130, MAP kinase, **protein kinase B**, and p70 S6 kinase in **cardiac** myocytes.

L4 ANSWER 3 OF 10 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1998:885066 SCISEARCH
THE GENUINE ARTICLE: 131UV
TITLE: Phosphatidylinositol 3-kinase transduces survival and hypertrophic signals via **Akt**/MAP kinase and p70 S6 kinase pathways in **cardiac** myocytes
AUTHOR: Oh H (Reprint); Kunasada K; Matsui H; YamauchiTakihara K
CORPORATE SOURCE: OSAKA UNIV, SCH MED, SUITA, OSAKA 565, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: CIRCULATION, (**27 OCT 1998**) Vol. 98, No. 17, Supp. [S], pp. 2432-2432.
Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436.
ISSN: 0009-7322.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 0

L4 ANSWER 4 OF 10 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1998:885065 SCISEARCH
THE GENUINE ARTICLE: 131UV
TITLE: Activation of gp130 inhibits doxorubicin induced cell death by Bcl-xL/caspace-3 interaction and PI 3-kinase/**Akt** pathway in **cardiac** myocytes
AUTHOR: Oh H (Reprint); Kunisada K; Funamoto M; YamauchiTakihara K
CORPORATE SOURCE: OSAKA UNIV, SCH MED, SUITA, OSAKA 565, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: CIRCULATION, (**27 OCT 1998**) Vol. 98, No. 17, Supp. [S], pp. 2431-2431.
Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436.
ISSN: 0009-7322.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 0

L4 ANSWER 5 OF 10 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999007021 MEDLINE
DOCUMENT NUMBER: 99007021 PubMed ID: 9792535
TITLE: Tyrosine phosphatase inhibitors, vanadate and pervanadate,

stimulate glucose transport and GLUT translocation in muscle cells by a mechanism independent of phosphatidylinositol 3-kinase and protein kinase C.

AUTHOR: Tsiani E; Bogdanovic E; Sorisky A; Nagy L; Fantus I G
CORPORATE SOURCE: Department of Medicine, Banting and Best Diabetes Centre, Mount Sinai Hospital, and University of Toronto, Ontario, Canada.

SOURCE: DIABETES, **(1998 Nov)** 47 (11) 1676-86.
Journal code: E8X; 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981110

AB Vanadate and pervanadate (pV) are protein tyrosine phosphatase (PTP) inhibitors that mimic insulin to stimulate glucose transport. To determine whether phosphatidylinositol (PI) 3-kinase is required for vanadate and pV, as it is for insulin, cultured L6 myotubes were treated with vanadate and pV. The two compounds stimulated glucose transport to levels similar to those stimulated by insulin; however, while PI 3-kinase activity and the increase in the lipid products PI 3,4-bisphosphate and PI 3,4,5-trisphosphate were inhibited by wortmannin after stimulation by all three agents--insulin, vanadate, and pV--wortmannin blocked glucose transport stimulated by insulin but not vanadate or pV. Vanadate and pV stimulated the translocation of GLUTs from an intracellular compartment to the plasma membrane; this stimulation was not blocked by wortmannin, but insulin-induced GLUT translocation was inhibited. Similar results were obtained in cultured H9c2 **cardiac** muscle cells in which wortmannin did not inhibit glucose transport or the vanadate-induced translocation of GLUT4 in c-myc-GLUT4 transfected cells. The ser/thr kinase PKB (**Akt**/PKB/RAC-PK) is activated by insulin, lies downstream of PI 3-kinase, and has been implicated in signaling of glucose transport. Insulin and pV stimulated PKB activity, and both were inhibited by wortmannin. In contrast, vanadate, at concentrations that maximally stimulated glucose transport, did not significantly increase PKB activity.

To determine the potential role of protein kinase C (PKC), L6 cells were incubated chronically with phorbol myristate acetate (PMA) or acutely with the PKC inhibitors calphostin C and bisindolylmaleimide. There was no inhibition of glucose transport stimulation by insulin, vanadate, or pV, and a combination of wortmannin and PKC inhibitors also failed to block the effect of vanadate and pV. In contrast, disassembly of the actin network with cytochalasin D blocked the stimulation of glucose transport by all three agents. In conclusion, vanadate and pV are able to stimulate glucose transport and GLUT translocation by a mechanism independent of PI 3-kinase and PKC. Similar to that by insulin, glucose transport stimulation by vanadate and pV requires the presence of an intact actin network.

L4 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1999:524207 BIOSIS

DOCUMENT NUMBER: PREV199900524207
TITLE: Activation of gp130 inhibits doxorubicin induced cell death
by Bcl-xL/caspase-3 interaction and PI 3-kinase/**Akt** pathway in **cardiac** myocytes.
AUTHOR(S): Oh, Hidemasa; Kunisada, Keita; Funamoto, Masanobu; Yamauchi-Takihara, Keiko
CORPORATE SOURCE: Osaka Univ. Med. Sch., Suita, Osaka Japan
SOURCE: Circulation, (**Oct. 27, 1998**) Vol. 98, No. 17 SUPPL., pp. I462.
Meeting Info.: 71st Scientific Sessions of the American Heart Association Dallas, Texas, USA November 8-11, 1998 The American Heart Association
. ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

L4 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1999:524208 BIOSIS
DOCUMENT NUMBER: PREV199900524208
TITLE: Phosphatidylinositol 3-kinase transduces survival and hypertrophic signals via **Akt**/MAP kinase and p70 S6 kinases pathways in **cardiac** myocytes.
AUTHOR(S): Oh, Hidemasa; Kunasada, Keita; Matsui, Hideo; Yamauchi-Takihara, Keiko
CORPORATE SOURCE: Osaka Univ. Med. Sch., Suita, Osaka Japan
SOURCE: Circulation, (**Oct. 27, 1998**) Vol. 98, No. 17 SUPPL., pp. I462.
Meeting Info.: 71st Scientific Sessions of the American Heart Association Dallas, Texas, USA November 8-11, 1998 The American Heart Association
. ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

L4 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1999:219629 BIOSIS
DOCUMENT NUMBER: PREV199900219629
TITLE: Recent progress in insulin signal transduction.
AUTHOR(S): Hei, Yong-Jiang (1)
CORPORATE SOURCE: (1) 56 Wood Acres Dr., E. Amherst, NY, 14051 USA
SOURCE: Journal of Pharmacological and Toxicological Methods, (**Oct., 1998**) Vol. 40, No. 3, pp. 123-135.
ISSN: 1056-8719.
DOCUMENT TYPE: General Review
LANGUAGE: English

L4 ANSWER 9 OF 10 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97445153 MEDLINE
DOCUMENT NUMBER: 97445153 PubMed ID: 9299480
TITLE: p70 S6 kinase is activated by sodium arsenite in adult rat **cardiomyocytes**: roles for phosphatidylinositol 3-kinase and p38 MAP kinase.
AUTHOR: Wang X; Proud C G
CORPORATE SOURCE: Research School of Biosciences, University of Kent at Canterbury, United Kingdom.
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, **(1997 Sep 8)** 238 (1) 207-12.
Journal code: 9Y8; 0372516. ISSN: 0006-291X.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971021
Last Updated on STN: 19990129
Entered Medline: 19971009

AB p70 S6 kinase (p70 S6k) is important in regulating a variety of cellular functions including mRNA translation and cell cycle progression and is activated by mitogens and hormones. Unexpectedly, we have found that, in adult rat **cardiomyocytes**, arsenite, which generally induces stress responses, markedly and rapidly activates p70 S6k. This activation of p70 S6k is completely blocked by rapamycin but only partially prevented by inhibitors of phosphatidylinositol 3-kinase. In trying to delineate the mechanism underlying this effect, we found that arsenite did not activate **protein kinase B**, JNK or MAP kinase, but did activate p38 MAP kinase in **cardiac** myocytes. A specific inhibitor of p38 MAP kinase (SB203580) partially attenuated the stimulation of p70 S6k by arsenite. These data indicate that the activation of p70 S6k by arsenite involves p38 MAP kinase and phosphatidylinositol 3-kinase but not PKB.

L4 ANSWER 10 OF 10 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 94025762 MEDLINE
DOCUMENT NUMBER: 94025762 PubMed ID: 8212714
TITLE: [Bioprosthesis degeneration in the aortic and mitral valve position. Results and problems from the cardiosurgical viewpoint].
Bioprothesendegeneration in Aorten- und Mitralposition. Ergebnisse und Probleme aus kardiochirurgischer Sicht.
AUTHOR: Antretter H; Cottogni M; Falbesoner C; Furtwangler W; Mair P; Falk M; Hutter J
CORPORATE SOURCE: Abteilung fur Herzchirurgie, I. Universitatsklinik fur Chirurgie, Innsbruck.
SOURCE: WIENER MEDIZINISCHE WOCHENSCHRIFT, **(1993)** 143 (11) 281-7.
Journal code: XOU; 8708475. ISSN: 0043-5341.
PUB. COUNTRY: Austria
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199311
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 19940117
Entered Medline: 19931122

AB 38 patients (20 men, 18 women) underwent reoperation between July 1989 to September 1992 because of degeneration of bioprosthetic valves implanted in aortic or/and mitral position. Retrospective analysis revealed a mean implantation time of 116.5 +/- 31.5 months for the bioprostheses (median 116). At reoperation 63.2% of them had a single valve replacement, 36.8% a more complex **cardiac** procedure (double or triple valve replacement, valve replacement and coronary bypass grafting). 50% (n = 9) of the reoperation cohort were symptomatic (NYHA III), 16 (42.1%) were serious symptomatic (NYHA IV). All deaths were NYHA IV preoperatively. Early mortality was 18.4% (n = 7). Mean age at the time of first operation

was 51 +/- 10.7, mean age at reoperation was 60.5 +/- 10.6. There was a significantly longer aortic clamp time (**AKT**, p = 0.0005) and bypass time (BPT, p = 0.0000) compared to first operation, also a significantly longer BPT of the deads confronted with the survivors (p = 0.0075). Bioprosthetic valves in mitral position were significantly longer implanted (p = 0.0416) than in aortic position. But there was no difference in implantation time of commercially available Carpentier-Edwards- or Ionescu-Shiley grafts. At reoperation we changed more than 95% of the degenerated valves to mechanical devices--corresponding to international tendencies. We discuss the early tissue degeneration of bioprosthetic valves and their increasing problem during reoperation.